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Dysmenorrhea

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Continuing Education Activity

Dysmenorrhea is defined as pain during the menstrual cycle. The pain is usually located in the lower abdomen and may radiate to the inner thighs and back. It is a very common gynecologic problem and can negatively impact a patient's life. Providing treatment options for patients affected by dysmenorrhea can significantly reduce the associated morbidity. There are various treatment options, some of which may be more or less helpful for an individual patient. This activity reviews the evaluation and management of dysmenorrhea or painful menstruation. It highlights the role of the interprofessional team in evaluating and treating patients with dysmenorrhea, as well as appropriately referring to subspecialty care when indicated.

Objectives:

- Identify the etiology of primary and secondary dysmenorrhea.
- Apply an evaluation process for primary and secondary dysmenorrhea.
- Differentiate the management options available for primary and secondary dysmenorrhea.
- Implement interprofessional team strategies for improving care coordination and communication to advance the recognition of and treatment options for dysmenorrhea to improve overall outcomes.

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Introduction

Dysmenorrhea is a Greek term for "painful monthly bleeding." [1] Dysmenorrhea can be classified as primary or secondary. Primary dysmenorrhea is recurrent lower abdominal pain that happens during the menstrual cycle and is not associated with other diseases or underlying pathology.[2] It is a diagnosis of exclusion. In contrast, secondary dysmenorrhea is associated with suspected or clinically identifiable pathology.[3] Dysmenorrhea is a common complaint among menstruating patients during their reproductive years. Dysmenorrhea may be associated with significant negative emotional, psychological, and functional health impacts.[4]

Primary dysmenorrhea classically begins within about 2 years of menarche or once ovulatory cycles have been established. It is more often a diagnosis made in adolescents and young adults. The cyclic pain starts within a few hours of the onset of menses and usually resolves within 72 hours. The pain is located midline in the pelvis and may radiate to the lumbar area of the back or upper legs.[5] It may be crampy and episodic and is usually similar in each menstrual cycle. Concomitant symptoms may include nausea, vomiting, headaches, dizziness, fatigue, and sleep difficulties.[6]

Etiology

Since the 1960s, many theories have been proposed to explain the etiology of dysmenorrhea. These theories include psychological, biochemical, and anatomical etiologies. The anatomical theory notes abnormal uterine position and abnormalities in the shape or length of the cervix. Zebitay et al, in their study, proposed a positive correlation between cervical length and the volume and intensity of dysmenorrhea.[7] According to multiple other studies, the biochemical theory has the most substantial evidence.[8]

Associated risk factors for dysmenorrhea include the following:

- Age (commonly) up to 30 years
- Smoking [9]
- Attempts to lose weight
- Higher or lower than normal body mass index
- Depression/anxiety
- Longer menstrual cycles
- Younger age at menarche
- Nulliparity
- History of sexual assault
- Previous cesarean section with incomplete uterine scar healing (uterine niche)
- Longer and heavier menstrual flow
- Family history of dysmenorrhea
- Disruption of social networks [10][6]

Primary Dysmenorrhea: Prostaglandins (PGs) are thought to be the main cause of dysmenorrhea.[11][12] Higher levels of PGs have been noted in the menstrual fluid and endometrial tissue of women with dysmenorrhea.[5] Endometrial shedding begins due to the decreasing hormone levels in the menstrual cycle. The time of endometrial shedding during the beginning of menstruation is when the endometrial cells release PGs. PGs cause uterine contractions, and the intensity of the cramps is proportionate to the amount of PGs released.[13][14] Uterine contractions cause tissue hypoxia and ischemia, which in turn cause pain and sometimes associated nausea and diarrhea.[5]

Secondary Dysmenorrhea: Secondary dysmenorrhea is menstrual pain due to an underlying disease, disorder, or structural abnormality within or outside the uterus.[15] It may affect women at any time after menarche. It can be a new symptom for females in their 30s or 40s. Secondary dysmenorrhea can be associated with varying intensities of pain and, at times, other symptoms such as dyspareunia, menorrhagia, intermenstrual bleeding, and postcoital bleeding. There are many common causes of secondary dysmenorrhea, including endometriosis, large cesarean scar niche, fibroids, adenomyosis, endometrial polyps, interstitial cystitis, pelvic inflammatory disease, and possibly the use of an intrauterine contraceptive system.[11][6][16] Up to 29% of women with dysmenorrhea may have endometriosis. When considering NSAID-resistant dysmenorrhea, up to 35% of patients may have endometriosis.[17] Adenomyosis is another

common underlying disease that is associated with secondary dysmenorrhea. Up to 3.8% of young women have reproductive tract anomalies, and both obstructive and non-obstructive anomalies may be associated with secondary dysmenorrhea.

Epidemiology

Dysmenorrhea is one of the most common gynecological concerns among all menstruating patients, regardless of age or race. It is a frequently identified cause of pelvic pain. The prevalence of dysmenorrhea can vary between 16% and 91% in individuals of reproductive age, with severe pain observed in 2% to 29% of individuals.[10] Agarwal et al showed the prevalence of dysmenorrhea to be 80% in adolescents. Of those affected adolescents, approximately 40% had severe dysmenorrhea.[18]

The symptoms associated with dysmenorrhea may include gastrointestinal symptoms such as nausea, bloating, diarrhea, constipation, vomiting, and indigestion. Irritability, headache, and lower back pain are prevalent among women presenting with primary dysmenorrhea. Tiredness and dizziness may also be associated.[19] In approximately 16% to 29% of women, dysmenorrhea is associated with significant impairment in quality of life.[10] Furthermore, 12% of monthly school and work activities may be missed due to absenteeism because of dysmenorrhea.[20]

Pathophysiology

The pathophysiology of primary dysmenorrhea is not fully understood. Nevertheless, the identified cause is believed due to the hypersecretion of PGs from the uterine inner lining. PGs cause pain by increasing uterine contractions and uterine pressure. Impaired uterine perfusion, ischemia, hypoxia, and metabolites from anaerobic metabolism may also play a role in the cause of pain.[6] The increase in collagenases, inflammatory cytokines, and matrix metalloproteinases in the endometrium is associated with decreased progesterone and estradiol with menstruation. The subsequent breakdown of endometrial tissue frees phospholipids, which are converted to arachidonic acid. The arachidonic acid is converted to prostacyclins, PGs, and thromboxane-2a via cyclooxygenase.[17] The products PG F2 alpha (PGF-2α) and PG E2 (PGE2) increase the uterine tone and cause high-amplitude contractions of the uterus.[21]

The expression of COX-2, a cyclooxygenase, is very high during menstruation, and this is what has driven the use of NSAIDs for treatment, as noted below.[17] Vasopressin has also been linked to primary dysmenorrhea. Vasopressin increases uterine contractility and can cause ischemic pain due to its vasoconstrictive effects.[3][22] Leukotrienes C4 and D4 levels are higher in patients with dysmenorrhea and appear to be associated with an increase in uterine contractions as well.[6] Furthermore, uterine contractility is more prominent during the first 2 days of menstruation, which is when dysmenorrhea is most frequent and severe.[23]

Endometriosis and adenomyosis are the most common causes of secondary dysmenorrhea in premenopausal women.[23]

History and Physical

A comprehensive history, along with an adequate physical examination, is essential to establish the diagnosis of dysmenorrhea. A history, including the location of pain, timing of onset, characteristics, duration, and associated symptoms like fatigue, headache, diarrhea, nausea, and vomiting, can help establish a diagnosis.[19]

In patients with primary dysmenorrhea, the physical examination findings are usually unremarkable. The pelvic exam demonstrates a normal-sized uterus that is nontender and mobile. Lack of abnormal discharge, adnexal masses, and uterosacral nodularity are typical findings in primary dysmenorrhea.[6]

Common findings that may indicate secondary dysmenorrhea include the following:

- Older age (older than years old) versus younger age, in which primary dysmenorrhea is more common
- Vaginal discharge that is whitish gray, mucopurulent, or has a foul odor (pelvic inflammatory disease concern)
- Friable cervix (sexually transmitted infection concern)
- Dysuria, dyspareunia, vaginismus, dyschezia, infertility, palpable nodularity, adnexal masses, or tenderness on pelvic exam [24]
- Heavy menstrual bleeding with a mildly enlarged symmetrical uterus (adenomyosis)
- Abnormal bleeding with an enlarged asymmetrical uterus (leiomyomas possible)
- Obstructive anatomical abnormalities or history of other congenital anomalies (concern for Müllerian anomaly)
- Pelvic masses (neoplasms, ovarian cysts, endometrioma) [25][26]

Evaluation

Primary dysmenorrhea may be diagnosed based on the typical history alone.[5] Pain begins just before or at the start of menstrual bleeding. It is cyclic and begins with the onset of ovulatory cycles, usually within 2 years of menarche. Pain peaks at 23 to 48 hours after the onset of bleeding and usually lasts no more than 72 hours.[6]

1. A pelvic examination is important for evaluating dysmenorrhea if the history of onset and duration of lower abdominal pain suggests secondary dysmenorrhoea or if the dysmenorrhea is not responding to medical treatment.[26] A pelvic examination is not necessary for an adolescent who is not sexually active and has a typical presentation of primary dysmenorrhea without additional symptoms.[5]
2. The use of ultrasound in the evaluation of primary dysmenorrhea has little benefit. However, ultrasound can be useful in differentiating the cause of secondary dysmenorrhea, including endometriosis, leiomyomas, Mullerian anomalies, and adenomyosis.[11][26] [5] Ultrasound is the preferred initial evaluation of the cause of secondary dysmenorrhea.
3. Patients who are at risk of sexually transmitted infections (STIs) or when pelvic inflammatory disease (PID) is suspected may need endocervical or vaginal swabs.[11][26]
4. If indicated, cervical cytology samples and/or HPV testing may be considered to rule out a suspected cervical malignancy.

5. Magnetic resonance imaging (MRI) or Doppler ultrasonography may be useful if torsion of the adnexa, adenomyosis, or deep pelvic endometriosis is suspected or if there are inconclusive findings on the ultrasound.[26] MRI is particularly helpful in diagnosing Müllerian anomalies but is not cost-effective as an initial screening tool.

6. Laparoscopy is usually reserved for women who desire fertility and have suspected endometriosis as a cause of secondary dysmenorrhea.

Treatment / Management

Treatment of dysmenorrhea is aimed at providing adequate pain relief to allow patients to perform most of their daily activities. Treatment for primary and secondary dysmenorrhea begins similarly. Patient education, reassurance, supportive therapy, and medical management are the initial baseline interventions recommended. If symptoms do not respond to initial treatments, an evaluation for potential causes of secondary dysmenorrhea may be warranted. Treatment strategies are divided into pharmacologic and nonpharmacologic treatments as below. Opioids and tramadol should not be regularly used to treat dysmenorrhea.

Nonpharmacological Treatment

Heat application, exercise, and diet: Baseline intervention starts with heat application and exercise. Heat applied to the lower abdomen may be as effective as NSAIDs and more effective than acetaminophen, with no associated side effects. This is the preferred initial therapy option by many patients.[27][28] Heat applied to the lower abdomen may be as effective as NSAIDs and more effective than acetaminophen, with no associated side effects. This is the preferred initial therapy option by many patients.[27][28] Evidence does support the role of regular exercise in the treatment of dysmenorrhea, but the specific type, duration, and frequency of exercise are not clear. Moderate exercise is recommended for all patients.[29]

Maintaining an active lifestyle and a balanced diet rich in vitamins and minerals is generally recommended for better health outcomes. In particular, such diet and lifestyle changes may be useful to reduce the intensity of dysmenorrhea.[30][31][32] Food supplements, complementary or alternative medicine such as plant-based therapy, and Chinese medicine are being used for the treatment of dysmenorrhea. However, they are not regulated by the FDA. Overall, there is insufficient evidence to recommend the use of any of the other herbal and dietary therapies.[33]

Spinal manipulation has shown no benefit in PG levels or pain associated with primary dysmenorrhea in at least 1 high-quality randomized controlled trial.[34]

Acupuncture, transcutaneous electrical nerve stimulation, and behavioral counseling: The effectiveness of acupuncture is supported by a few studies that lack active comparisons and sound methodological techniques.[35][36][37] Transcutaneous electrical nerve stimulation and behavioral counseling may be used as second-line nonpharmacological treatments.[17]

Pharmacological Treatment

Nonsteroidal anti-inflammatory drugs (NSAIDs) are considered to be first-line pharmacologic treatment for dysmenorrhea. NSAIDs have been shown to be effective in the treatment of dysmenorrhea in comparison to placebo.[38] NSAIDs exert their benefit by inhibiting cyclooxygenase enzymes, thereby blocking PG production.[39] Use of an NSAID on a scheduled

basis starting 1 to 2 days prior to the onset of pain has been shown to work better than when an NSAID is used on an as-needed basis.[6]

In a systematic review comparing different NSAIDs to placebo in the treatment of dysmenorrhoea, Marjoribanks et al concluded that overall, no NSAID is safer or more effective than others.[40] A meta-analysis of 70 studies showed that flurbiprofen and tiaprofenic acid were superior for treating dysmenorrhea; however, tiaprofenic acid is not available in the United States.[6] Unfortunately, there is evidence that approximately 20% of patients with dysmenorrhea do not respond to treatment with NSAIDs. This has been termed NSAID-resistant dysmenorrhea.[17][41]

Fenamates, like mefenamic acid, may have slightly better efficacy than the phenyl propionic acid derivatives (ibuprofen, naproxen) because fenamates have dual action of blocking the production of PGs and inhibiting their action.[42][43] Mefenamic acid 500 mg with the start of menses or pain followed by 250 mg every 6 hours for up to 3 days may be helpful. One study recommended ibuprofen and fenamates as preferred in terms of safety and efficacy.[44] Ibuprofen 800 mg every 8 hours or naproxen 440 mg to 550 mg as an initial dose followed by 220 mg to 550 mg every 12 hours on a scheduled basis have been recommended. NSAIDs are more effective as compared to paracetamol (acetaminophen). However, paracetamol is still a valid alternative when NSAIDs are contraindicated. Celecoxib, a COX-2 selective NSAID, was used for the treatment of dysmenorrhea in the past but is no longer recommended due to its cardiovascular side effects. Black box warnings regarding the risk of serious adverse events must be heeded.[45] COX-2 selective NSAIDs have also been linked to delayed ovulation since PGs are needed for ovulation.[46][47]

Acetaminophen is a pain reliever that may be an alternative for patients who cannot take NSAIDs. Acetaminophen with pamabrom or caffeine (both diuretics) has shown reduced pain from dysmenorrhea.[48][49][50]

Hormonal contraception with estrogen and progesterone in the form of pills, patches, or vaginal rings is reportedly effective in reducing dysmenorrheic pain as compared to placebo.[51][52][53][54] Any available combination pill provided similar pain relief with no difference in efficacy. Other studies have argued against the effectiveness of combined hormonal contraceptives as a treatment for dysmenorrhea due to small sample sizes and limited comparative data.[55][56] A combination estrogen and progesterone contraceptive will limit endometrial growth as well as inhibit ovulation. Over time, as the endometrium thins, menses become lighter, and fewer uterine contractions occur with menses. This, in turn, decreases pain with menses. Combination birth control pills (COCs) also work by decreasing the production of PGs and leukotrienes.[38] Low levels of PGs are noted in the menstrual fluid of women on COCs. Contraceptive pill users appear to have significantly lower rates of dysmenorrhea and need fewer additional analgesics for treatment.[13] Continuous regimens of hormonal contraception, wherein the placebo pills are not taken, are even more beneficial in treating dysmenorrhea than cyclic therapy.[17]

Progestin-only contraception, including pills, implants, IUDs, or intramuscular injections, is suitable particularly for patients with secondary dysmenorrhea related to endometriosis. Their effectiveness as a treatment for primary dysmenorrhea is not evident.[57][58][59] Progesterone works by causing atrophy of the endometrial lining and by inhibiting ovulation. Dienogest and norethindrone acetate are oral formulations of progesterone that are

specifically recommended as primary treatment for secondary dysmenorrhea due to endometriosis.[17]

Gonadotropin-releasing hormone agonists and antagonists may be offered as second-line pharmacologic treatment. GnRH agonists include nafarelin, leuprolide acetate, and goserelin. The GnRH antagonist is elagolix. These are effective treatments for dysmenorrhea caused by endometriosis, but cost and adverse effects may limit the use of these therapies, especially without the use of add-back estrogen and progesterone therapy. The American Society of Reproductive Medicine recommends the use of gonadotropin-releasing hormone agonists to treat dysmenorrhea after the laparoscopic diagnosis of endometriosis. However, these medications are not considered long-term therapies.[17]

Aromatase inhibitors may be used to treat secondary dysmenorrhea as they induce amenorrhea. However, adverse effects, including loss of bone mineral density, may prohibit use, especially without concomitant estrogen and progesterone add-back therapy.

A vasodilator, sildenafil citrate, reduces pain with primary dysmenorrhea. It promotes smooth muscle relaxation in the uterus. Nitric oxide donor drugs like transdermal nitroglycerin or glyceryl trinitrate patches (0.1 mg) similarly relax smooth muscle. Headaches and feeling light-headed are significant side effects of vasodilators, and these medications are typically not used for first-line treatment of dysmenorrhea.[6] Further studies are needed to determine the utility of treatment with vasodilators for dysmenorrhea.[17]

Calcium channel blockers, such as nifedipine 20 mg to 40 mg, inhibit uterine contractions and decrease pain from dysmenorrhea. However, headache, tachycardia, and flushing as adverse effects may limit use.[17]

Vasopressin/oxytocin receptor agonists have been studied in the treatment of dysmenorrhea since these hormones are known to stimulate myometrial contractions. Atosiban and SR49059 have both been studied for NSAID-resistant dysmenorrhea; however, no conclusions on the timing, effectiveness, or method of administration have yet been made.[17]

Antispasmodics have been studied in the treatment of dysmenorrhea since muscle spasm has been implicated as a cause of dysmenorrhea. Globally, hyoscine butylbromide has been used to treat dysmenorrhea due to its anticholinergic effects on muscarinic receptors, resulting in smooth muscle relaxation. A similar medication, hyoscyamine sulfate, is available in the United States, but it is not FDA-approved for use for dysmenorrhea. In a randomized controlled trial, the use of an antispasmodic medication along with an NSAID gave better results than the use of an NSAID alone.[17]

Magnesium has both muscle-relaxing effects and vasodilator properties and has thus been shown to reduce pain from dysmenorrhea. Lack of formulation and dosage recommendations have resulted in the conclusion that magnesium should be used in combination with other therapies, not alone in the treatment of dysmenorrhea.[6]

Surgical Options

Surgical options should only be used if there is not a satisfactory response to trials of medical management.

Laparoscopy is the next step in the evaluation and treatment of dysmenorrhea if the patient has a high likelihood of pathology as the underlying cause of the dysmenorrhea and if relief has not been accomplished within 3 to 6 months of initial pharmacologic treatment. The goal of surgery

is to resect endometriotic implants. Postoperative suppression of any residual or microscopic endometriosis is recommended with either progesterone alone, combination birth control, or GnRH agonist therapy.

Endometrial ablation is an option to consider in patients with heavy menstrual bleeding who are done with childbearing if medical management has not achieved satisfactory results.

Hysterectomy may be offered as a last resort to women who have failed all other possible treatment modalities. Removal of the ovaries may be considered depending on the etiology of the dysmenorrhea, the age of the patient, and the risk of needing additional surgery for removal of ovaries in the future.

Nerve transection procedures, including laparoscopic uterine nerve ablation and presacral neurectomy, are not recommended except possibly in select cases of patients with refractory midline pelvic pain. Further research is warranted on these procedures.[17] Pain has been noted to recur when nerve regeneration occurs postoperatively. Adverse effects are significant and may include pelvic organ prolapse, constipation, and urinary dysfunction.[6]

Differential Diagnosis

The differential diagnosis of dysmenorrhea is broad. It can be categorized as gynecological conditions and non-gynecological conditions: [60]

Gynecological Conditions

- Endometriosis
- Obstruction of the reproductive tract: Imperforate hymen, transverse vaginal septum, vaginal agenesis, OHVIRA syndrome (uterus didelphys with obstructed hemivagina and ipsilateral renal agenesis), and cervical stenosis
- Functional and nonfunctional adnexal cysts
- Adnexal torsion (this usually does not present with cyclic pain with menses)
- Adenomyosis
- Pelvic inflammatory disease/sexually transmitted infections
- Endometrial polyps
- Asherman syndrome
- Ectopic pregnancy
- Chronic pelvic pain
- Membranous dysmenorrhea: a very uncommon cause of colicky pain from uterine contractions with resultant shedding of the endometrium in one piece, which retains the shape of the uterus [61]

Non-Gynecological Conditions (gastrointestinal, urological, and musculoskeletal)

- Irritable bowel syndrome
- Urinary tract infections

- Interstitial cystitis
- Musculoskeletal causes: abdominal wall muscles, abdominal wall fascia, pelvic and hip muscles, sacroiliac joints, and lumbosacral muscles

Pertinent Studies and Ongoing Trials

Various phenotypes of dysmenorrhea likely exist, each with their underlying causes. The use of breakthrough pain management in the treatment of dysmenorrhea and tracking of this along with menstrual pain may be helpful in the future as pertinent studies of NSAID-resistant treatment of dysmenorrhea evolve.[17]

Prognosis

An initial medical treatment method is begun and continued for 2 to 3 months before a reassessment of symptoms is performed. If symptoms improve but are still present, a second treatment method may be added. If minimal to no response is noted, a change of treatment with discontinuation of the initial treatment may be offered. Patients are continued on treatment for an additional 3 months and then reassessed. If an adequate response is not obtained, evaluation for an underlying cause of secondary dysmenorrhea may be initiated. A multidisciplinary pelvic pain evaluation with physical therapy input and treatment may also be considered.

Dysmenorrhea may have a significant impact on patients' day-to-day lives. Such impact is reflected in the rates of absenteeism from school or work. Dysmenorrhea may also limit a patient's participation in sports or social events. Furthermore, there are associated emotional stressors associated with dysmenorrhea. In the United States alone, dysmenorrhea is estimated to be the cause of missing roughly 140 million working hours per year, a public health matter that has a significant economic impact.[62]

With the use of recommended treatment options, the prognosis for primary dysmenorrhea is generally good. Mild and moderate dysmenorrhea usually responds well to NSAIDs. Severe dysmenorrhea may still respond to NSAIDs but may require higher doses or combination/adjuvant therapy. In the case of persistent dysmenorrhea, secondary causes of dysmenorrhea should be investigated. The prognosis of secondary dysmenorrhea depends on the etiology, type, location, and severity of the cause.

Complications

Primary dysmenorrhea complications can be summarized by the intensity of the pain and how it affects the patient's well-being and disrupts daily activities. Since primary dysmenorrhea is not linked to any pathology or disease, there are no additional known complications.

In contrast, secondary dysmenorrhea complications vary depending on the etiology. Complications may include infertility, pelvic organ prolapse, heavy bleeding, and anemia, to name a few.[23][63]

Deterrence and Patient Education

Balanced, healthy nutrition and regular exercise reduce the severity of dysmenorrhea.[64] It is important to educate and create awareness among young patients regarding the importance of

properly balanced nutrition for reducing the pain from dysmenorrhea. Some vitamins and healthy dietary modifications have been associated with reduced menstrual pain.[31][65][66]

Regular physical activity is effective in reducing dysmenorrhea. Exercise acts as a nonspecific analgesic by improving pelvic circulation and stimulating the release of β-endorphins.

The primary goal of treatment is to reduce pain and improve the quality of life in patients suffering from dysmenorrhea. Medical and procedural treatments should be used appropriately to allow affected patients to perform their day-to-day activities without missing significant amounts of school or work. Patients should be counseled to follow up with their healthcare provider when dysmenorrheic symptoms are bothersome and not well-controlled.

Enhancing Healthcare Team Outcomes

In managing dysmenorrhea, a multifaceted approach involving a diverse team of healthcare professionals is essential to ensure patient-centered care and optimal outcomes. Physicians, advanced care practitioners, nurses, pharmacists, pelvic physical therapists, and other healthcare experts must possess a comprehensive skill set, including a deep understanding of gynecological health and pain management.

The interprofessional team's strategy should prioritize evidence-based, personalized treatment plans that address the unique needs and preferences of each patient while adhering to ethical guidelines that respect patient autonomy and informed consent. Patients should be counseled appropriately regarding the treatment options for dysmenorrhea as well as the potential complications associated with secondary dysmenorrhea. The management of a patient with dysmenorrhea depends on the severity of symptoms and the responsiveness to treatment. Improved outcomes with less interference in activities of daily living are the goal.

Clinicians are responsible for providing empathetic and culturally sensitive care that promotes patient safety and minimizes potential adverse effects or complications. Effective interprofessional communication is paramount to ensure a seamless flow of information and coordinated care, as it enables the team to collaboratively tailor treatments and interventions to the individual patient. By uniting their skills, strategy, ethics, responsibilities, and communication efforts, the interprofessional team can enhance patient-centered care, improve outcomes, boost patient safety, and optimize team performance in managing dysmenorrhea.

Review Questions

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